Medicine Insights

Review

Uterine Artery Hemodynamic Variations among TCM Syndrome Types in Early-Gestation Subchorionic Hematoma

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Abstract: Subchorionic hematoma (SCH) is a frequent first-trimester obstetric complication marked by separation between chorionic membrane and uterine wall, with blood pooling surrounding the gestational sac within 12 gestational weeks. Clinical manifestations include abdominal discomfort. Severe cases may result in threatened miscarriage, inevitable abortion or embryonic arrest, endangering maternal-fetal safety. Ultrasound-derived uterine artery hemodynamic indices serve as objective measurements for uteroplacental circulation and are widely adopted for SCH risk evaluation and prognostic assessment. In traditional Chinese medicine (TCM), SCH corresponds to Tailou and Dongtai Bu'an. Its pathogenesis is closely linked to kidney deficiency, qi-blood insufficiency and heat-blood patterns, each presenting distinct pathological mechanisms and clinical features. This review summarizes recent domestic and international literature, covering TCM differentiation rules for SCH, clinical utility of uterine artery hemodynamic indicators, correlations between TCM patterns and hemodynamic profiles, and underlying mechanisms. It aims to offer clinical references for integrated Chinese-Western management and support individualized, precise management for SCH.

Keywords: early-gestation subchorionic hematoma; TCM syndrome pattern; uterine artery hemodynamic index; clinical correlation

1. Research Background and Significance

SCH mostly occurs in patients experiencing threatened miscarriage. Partial detachment of uterine trophoblasts triggers subchorionic hemorrhage, and accumulated blood between chorionic membrane and decidua basalis forms hematoma, which appears as perigestational intrauterine effusion under B-ultrasound [1]. Published data demonstrate that SCH raises miscarriage risk; larger hematoma size correlates with higher likelihood of pregnancy loss. It also increases risks of preterm birth and placental abruption, requiring timely clinical intervention [2].

SCH etiology remains incompletely clarified. Two mainstream pathogenic theories include chorion-decidual vascular rupture and elevated decidual vascular permeability leading to exudative bleeding [3]. The former responds well to irritation avoidance and uterine contraction inhibition, whereas the latter is largely associated with autoimmune disturbance and coagulation defects. Ultrasonography acts as the primary diagnostic tool, visualizing hematoma location, dimension and morphology and enabling serial follow-up of lesion changes. Though MRI improves diagnostic accuracy, high expense restricts its routine clinical use [4].

Transvaginal ultrasound detects uterine artery hemodynamic indicators including pulsatility index (PI), resistance index (RI), and systolic-diastolic velocity ratio (S/D)[5]. These metrics reflect uterine vascular resistance and tissue perfusion. Physiologically in early pregnancy, uterine arteries dilate as implantation and placentation proceed; vascular resistance gradually drops, PI, RI and S/D decrease, and early-diastolic notches

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disappear to guarantee placental blood supply. For SCH patients, local bleeding induces uterine arterial spasm, elevated vascular resistance and insufficient perfusion. PI, RI and S/D values rise obviously, accompanied by higher positive rates of early-diastolic notches. The magnitude of index abnormality positively correlates with hematoma scale, bleeding duration and final pregnancy outcomes. Therefore, hemodynamic markers are widely used for illness assessment, curative effect monitoring and prognosis prediction for SCH patients.

Current Western-medical interventions mainly consist of bed rest, progesterone-based tocolysis and hemostatic supportive therapy. For patients with slow hematoma resorption or repeated episodes, single Western-drug treatment often yields unsatisfactory effects, creating demand for more personalized therapeutic schemes. Ancient TCM monograph Fu-ren-gui described four core symptoms of Tailou and Dongtai Bu'an: abdominal pain, lumbosacral soreness, bearing-down sensation and vaginal bleeding. Without proper treatment, these symptoms may progress to miscarriage or habitual abortion. TCM scholars believe damaged Chong-Ren meridians and unstable fetal essence constitute core pathogenesis. Blood stasis is recognized as a critical pathogenic factor [6]. Classic texts such as Synopsis of Prescriptions of the Golden Chamber and Treatise on Blood Syndromes recorded that extravasated blood inside uterine cavity belongs to blood stasis. Stagnant blood blocks Chong-Ren vessels, prevents nutrient-blood from nourishing the fetus and triggers vaginal bleeding or even miscarriage. Apart from blood stasis, kidney deficiency, qi-blood insufficiency, heat-blood disturbance and physical trauma are major risk contributors for SCH onset.

2. Domestic and International Research Overview

2.1. Modern-Medical Understanding of Subchorionic Hematoma

International SCH research started in the 1980s, focusing on risk factors and the connection between ultrasonic features and gestational outcomes. Heller et al [7]. analyzed 434 ultrasound scans of viable singleton pregnancies from 6 to 11 gestational weeks. Their findings revealed that when the hematoma-to-gestational-sac ratio exceeds 50 %, miscarriage risk reaches 23 %. Early SCH onset and advanced maternal age (over 35 years old) are adverse prognostic signals. Subplacental-located hematomas produce poorer outcomes than marginal hematomas [8]. SCH incidence reaches 17 % among early-pregnancy threatened-miscarriage patients with vaginal bleeding [9].

SCH arises from multiple risk causes: coagulation disorder, immune dysfunction, assisted reproductive technology (ART), gestational medication and reproductive-tract infection. Hypercoagulable status raises adverse-pregnancy risks. Elevated D-dimer and platelet-aggregation rate predict poor prognosis for SCH patients combined with recurrent spontaneous abortion (RSA), suggesting anticoagulant measures may benefit such population [10]. Compared with healthy gravidas, SCH patients show higher fibrinogen, D-dimer and homocysteine levels, while protein-S activity declines [11]. Protein S participates in anticoagulation by activating protein C to degrade pro-coagulant factors [12]. Excess homocysteine triggers endothelial injury and hypercoagulability, facilitating hematoma formation. Coagulation abnormalities may induce either hemorrhagic or thrombotic SCH [13].

Immune dysregulation is another important cause. Maternal immune tolerance is essential for fetal survival. Autoantibody positivity increases threatened-miscarriage probability. High-titer antinuclear antibodies serve as SCH risk markers [14]. Imbalanced Th1/Th2 cytokine profiles are observed in SCH patients, with Th1-dominant inflammatory responses suppressing embryo implantation and trophoblast development [15].

SCH occurs more frequently in IVF-achieved pregnancies. Frozen-thawed embryo transfer, multiparity and blastocyst transfer are positively associated with SCH incidence [16,17]. Intrauterine surgical injury to endometrium also increases susceptibility. Recent studies indicate estrogen level is not directly linked to SCH risk in medicated embryo-

transfer cycles [18]. Lower-genital-tract infection aggravates hematoma enlargement. Pathogens such as Ureaplasma urealyticum and Chlamydia disrupt immune balance; inflammatory mediators damage vascular endothelium and activate coagulation cascades [19,20]. Enhanced peripheral oxidative stress is detected in SCH patients after IVF-ET, implying potential pathogenic relevance [21]. SCH patients demonstrate reduced hCG and progesterone concentrations; lower hormone levels correspond to higher risks of spontaneous abortion, fetal death and preterm labor [22].

Multiple studies verify higher PI, RI and S/D values among SCH patients versus normal pregnant women, and abnormal hemodynamic indices correlate with unfavorable pregnancy results. Standard clinical guidelines for SCH remain absent. Asymptomatic minor SCH receives expectant observation. For moderate-to-large SCH, clinical goals include symptom relief and hematoma absorption through uterine contraction inhibition, hemostasis and anti-infection treatment [22,23]. Dynamic hemodynamic monitoring helps evaluate therapeutic responses and adjust clinical medication.

2.2. TCM Perspectives on Subchorionic Hematoma

Different TCM schools have summarized SCH pathogenesis. Lingnan-Luoshi Gynecology school holds that spleen-kidney deficiency combined with blood stasis is the core mechanism [24]. Congenital kidney essence and acquired spleen qi jointly maintain gestational stability. When spleen-kidney function declines, extravasated-blood stasis obstructs uterus and Chong-Ren meridians, so the fetus loses adequate nourishment. Professor Li Ke-qin proposed yin-deficiency internal heat and blood-stasis-induced fetal restlessness, especially common in ART-conceived pregnancies, which is related to maternal age, dietary habits and emotional stress [25]. Professor Kuang Ji-lin emphasized that congenital weakness, over-strain-caused spleen-kidney injury and qi failing to control blood contribute to SCH, with internal blood stasis as an important intermediate pathological product [26]. Professor Jin Zhi-chun pointed out that spleen-kidney deficiency and blood stasis interact and form vicious pathological cycles [27]. Director Wang Jun-ling considered kidney deficiency as the root cause, and intrauterine retained extravasated blood further aggravates continuous vaginal bleeding [28]. Some scholars put forward the "triple-essence insufficiency" theory: deficiency of kidney essence, spleen essence and fetal essence constitutes the root cause, while blood stasis represents external manifestation [29].

Common therapeutic strategies include tonifying spleen-kidney and activating blood circulation to stabilize gestation. Representative prescriptions include Shoutai Pills, Antai Decoction, Zishen Yutai Pills and Si-wu Decoction [30]. According to Gynecology of Traditional Chinese Medicine textbook, SCH is classified into three major patterns:

Kidney-deficiency pattern: Scant pale dilute vaginal bleeding during gestation, lower-abdominal distending discomfort, fatigue, poor appetite, loose stool, lumbosacral soreness, pale enlarged tongue with tooth marks, thin-white coating, deep-slow pulse.

Qi-blood-insufficiency pattern: Scant pale thin vaginal bleeding, dull lower-abdominal pain, sallow complexion, palpitations, shortness of breath, dizziness, lassitude, pale tongue, thin-white coating, fine-weak pulse.

Heat-blood pattern: Scant bright-red or dark-red vaginal bleeding, burning-like lower-abdominal pain, irritability, dry-bitter mouth, dry stool, red tongue with yellow coating, slippery-rapid pulse.

3. Uterine-Artery Hemodynamic Indices: Detection and Clinical Value

3.1. Detection Procedures

One experienced sonographer completed all transvaginal ultrasound examinations using GE Voluson E6 color-Doppler equipment (transducer frequency:5-9 MHz). Patients emptied bladder and took lithotomy position and kept stable breathing. The transducer was gently placed in posterior vaginal fornix without extra pressure. At 1-2 cm lateral to cervicouterine junction, bilateral uterine artery trunks were identified under color-Doppler mode. Doppler sampling gate was placed 1 cm below arterial bifurcation. The

ultrasonic beam was kept parallel to blood vessel. Spectra of 3-5 cardiac cycles were collected for measuring S/D, RI and PI, and waveform abnormalities were recorded.

3.2. Clinical Application

In obstetric practice, elevated PI reflects insufficient placental perfusion and relates to fetal growth restriction and pre-eclampsia [31]. RI mirrors distal vascular resistance, and S/D ratio evaluates vascular resistance affecting fetal oxygen supply [32,33]. Combined use of these three indices improves diagnostic reliability. In SCH patients, impaired spiral-artery remodeling leads to persistently high vascular resistance, manifesting as elevated PI, RI and S/D [34].

Hematoma resorption and gestational maintenance serve as core therapeutic targets. Declined RI, PI and S/D together with increased PSV and EDV after treatment indicate improved uterine circulation, which facilitates stagnant-blood dissipation and benefits trophoblast function. Different etiologies produce distinct hemodynamic features: pre-thrombotic-state-related SCH shows obvious vascular-resistance elevation, whereas hormone-deficiency-caused SCH may present decreased PSV and EDV. Serial hemodynamic monitoring supports therapeutic evaluation and timely regimen adjustment to improve treatment precision.

4. Summary

Early-gestation SCH is a common threat to maternal-fetal health. TCM syndrome-differentiated therapy exhibits unique advantages, and uterine-artery hemodynamic indices provide objective evidence for uteroplacental circulation assessment. Existing research demonstrates significant correlations between TCM patterns and hemodynamic features: kidney-deficiency-blood-stasis pattern shows prominent vascular-resistance rise; kidney-deficiency-heat-blood pattern presents moderate resistance increase; spleen-kidney deficiency and qi-blood-insufficiency patterns mainly manifest as perfusion shortage; simple kidney-deficiency pattern combines mild resistance elevation and hypoperfusion.

Such correlations deliver quantitative objective basis for TCM pattern standardization, guide individualized precise treatment, raise tocolysis success and hematoma-absorption rates, and support risk stratification and prognostic judgment. Current research still has defects: inconsistent syndrome criteria, limited sample volume and insufficient mechanism research. Future multi-center large-sample investigations and molecular-level exploration will further clarify the relationship between TCM patterns and hemodynamic changes in SCH. It will strengthen evidence for integrated Chinese-Western treatment, realize precise differentiation and targeted intervention, and finally optimize maternal-fetal clinical outcomes.

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